Refer to: Chater N, Mani J, Tonnemacher K: Superficial temporal artery bypass in occlusive cerebral vascular disease. Calif Med 119:9-13, Aug 1973

In carefully selected cases, superficial temporal artery bypass appears to be a promising and useful method for bringing about an improved collateral supply in problems of occlusive cerebral vascular disease. Such a procedure appears to improve the quality of life.

Superficial Temporal Artery Bypass in Occlusive Cerebral Vascular Disease

NORMAN CHATER, MD, JOHN MANI, MD, and K. TONNEMACHER, BS
San Francisco

IT IS WELL RECOGNIZED that stroke is a leading cause of death and disability in the United States.[1,2] It is third in frequency after heart disease and cancer as a cause of suffering in this country.[1] Approximately 200,000 new strokes occur per year, and this statistic may be a conservative estimate.[2] Two million people in America are at present disabled and unemployable as a result of this disease process.[3] Two-thirds are under 65 and in the so-called pre-retirement or productive years.[4] After the onset of a fixed stroke, rehabilitation may be extremely difficult[5] and the cost (in loss of human productivity) has been estimated at three billion dollars a year excluding acute hospitalization.[3] The seriousness of the problem is therefore apparent.

Natural History of Cerebral Vascular Disease and Arteriographic Investigation

Patients who have transient ischemic attacks (TIA's) have an 18 to 60 percent chance of having a completed stroke within a year.[6-8] One of the aims of stroke therapy, therefore, is to prevent such a catastrophe and, if possible, to improve the quality of life.[2] In a series of 100 consecutive patients presenting with TIA's (or TIA's with a mild completed stroke), 33 percent of those studied by arteriography were found to have significant obstructive lesions classified as "inaccessible or inoperable" by general vascular techniques.[9,11] Arteriographic investigations from the National Cooperative Study revealed 6 percent of the patients had purely inaccessible surgical lesions and an additional 33 percent had inaccessible combined with accessible lesions, the so-called "tandem lesion."[10] This is to be expected, as cerebrovascular artherosclerotic disease is a multifocal process. Other arteriographic studies have found 33 percent of obstructive lesions to be intracranial.[19] An additional 5 to 8 percent of patients studied for cerebrovascular symptoms were shown to have old complete internal carotid occlusions, which, at present, are considered inoperable by vascular surgeons because of the poor success rate in reopening the vessel.[10,11] At the same time, vascular surgeons are loath to operate on an acute internal carotid occlusion for fear of precipitating a hemorrhagic infarct.[13] Thus, some 12 percent

From the Department of Neurosurgery, Ralph K. Davies Medical Center, San Francisco.
Submitted January 10, 1973.
Reprint requests to: N. L. Chater, MD, Director, Microsurgical Unit, Ralph K. Davies Medical Center, 45 Castro Street, San Francisco, CA 94114.

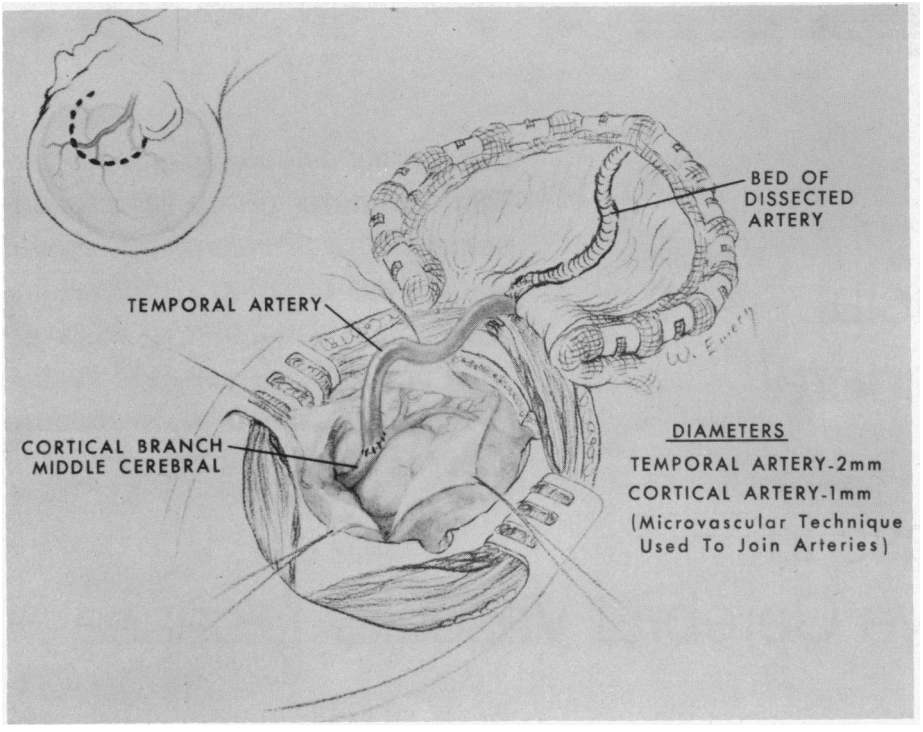

Figure 1. — Anastomosis, superficial temporal artery and branch of middle cerebral artery (collateral augmentation procedure).

of patients studied by arteriography may be found to have lesions that are "inoperable" by present conventional surgical techniques.[10,11] If the statistics are accurate, this potentially amounts to some 20,000 persons a year.

Surgical Procedure and Theoretical Considerations

For the bypass operation, the superficial temporal artery (average diameter 2 mm) is dissected free from the scalp and a burr hole or small craniectomy is fashioned of sufficient size to expose a suitable cortical artery, 1 mm in diameter or larger. Such arteries may now be found with high reliability.[14] An end-to-side anastomosis between the superficial temporal artery and the appropriate cortical artery is then performed with the operating microscope, using micro-instrumentation under 16 to 25 power magnification (Figure 1). The major obstruction may be bypassed in this manner[15,16] and an additional collateral blood supply provided for ischemic areas.[17,18] As "hemodynamic" cerebrovascular insufficiency symptoms are the result of insufficient collateral supply rather than the block itself,[19,20] such a surgical procedure may alleviate the symptoms and signs of cerebral ischemia.[17,19] Whether such a procedure can reverse pre-existing cerebrovascular neurological defects partially or completely is still open to considerable debate.[11,20] Some pathological studies have suggested, however, that at least minor deficits theoretically could be improved with the restoration of normal perfusion pressures to the areas of marginal flow.[21] At present there is no clinical method of determining in advance which neurons are nonfunctioning because of anoxia and which neurons are, in fact, dead. There is considerable experimental evidence, however, suggesting that neurons may live and tolerate anoxia without necrosis for considerable periods.[22]

Illustrative Cases

CASE 1. *Left Internal Carotid Occlusion and Right Carotid Siphon Stenosis with* TIA's

A 63-year-old, right-handed salesman was evaluated for recurrent attacks of left-sided numbness, weakness and dysarthria over a period of about six months. Twelve years previously he had had an attack of right-sided numbness and had completely recovered. Results of physical examination were within normal limits. Arteriograms revealed a complete left internal carotid occlusion with collateral supply through the ophthalmic system and 90 percent stenosis of the right carotid siphon. A right superficial temporal artery-cortical artery bypass was performed. The patient was asymptomatic thereafter and follow-up arteriograms six months later showed well functioning collateral vessels (Figure 2).

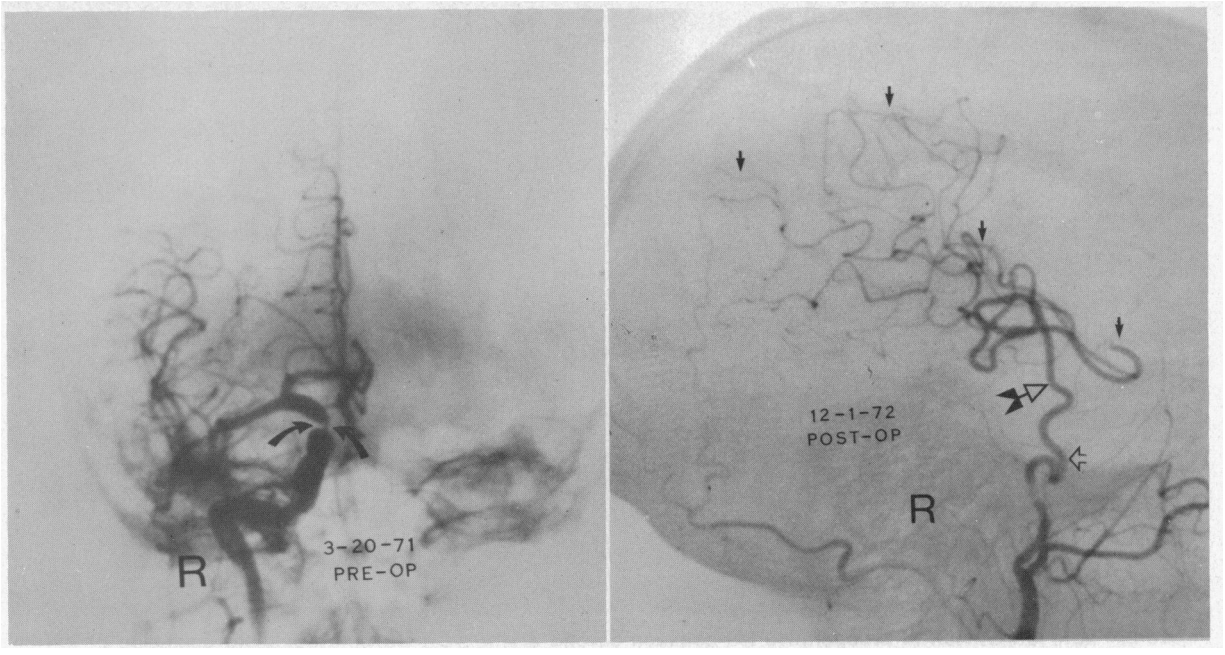

Figure 2.—Left, preoperative right common carotid arteriogram, antero-posterior view, showing severe carotid siphon stenosis (curved black arrows). **Right,** postoperative lateral view shows dilated superficial temporal artery (open arrow), point of anastomosis (black and white arrow), and middle cerebral artery filling (small black arrows).

CASE 2. *Left Middle Cerebral Stenosis with* TIA*'s*

A 64-year-old, retired, right-handed baker was investigated for two attacks of right-sided weakness, numbness and aphasia. Physical examination was unremarkable. Arteriograms showed 90 percent stenosis of the left middle cerebral artery with poor collateral vessels. Left superficial temporal artery-cortical artery anastomosis was performed. Postoperatively the patient remained asymptomatic. Follow-up arteriograms at six months showed a patent functioning anastomosis.

CASE 3. *Left Internal Carotid Artery Occlusion with* TIA*'s.*

A 59-year-old right-handed construction worker had quit working because of recurrent attacks of right-sided dysphasia, with numbness and weakness of the right arm and leg. Physical examination revealed moderate weakness of the right upper extremity and face with mild dysphasia and dysarthria. Arteriograms showed complete occlusion of the left internal carotid artery with poor collaterals from the left posterior communicating artery and right anterior communicating artery. Left superficial temporal-cortical artery anastomosis was carried out. The patient had no further attacks and on physical examination after the operation the right-sided weakness and dysphasia was minimal. Arteriograms four months after operation showed a patent anastomosis with good filling of the middle cerebral artery (Figure 3).

CASE 4. *Right Internal Carotid Artery Occlusion with Progressive Loss of Neurological Function*

A 54-year-old, Japanese clerk was admitted on suspicion of brain tumor because of progressive clumsiness over a period of two months, numbness and inability to distinguish objects in his left hand. On physical examination clumsiness of the left hand, decreased sensation to pinprick and severe astereognosis of the left upper extremity were noted. Arteriograms showed right internal carotid artery occlusion and precarious filling of collateral vessels through a small right posterior communicating artery. Right superficial temporal-parietal anastomosis was performed and a week later arteriograms showed a patent anastomosis with enlargement of the superficial temporal artery to approximately three time its preoperative diameter (Figure 4). In a short follow-up at the time of this report, astereognosis had diminished, as had sensory loss and clumsiness of the left hand.

CASE 5. *Left Internal Carotid Occlusion with Severe Cerebrovascular System Deficit*

A 44-year-old, right-handed, government agent had had two transient attacks of right-sided weakness and aphasia, with recovery, followed by an

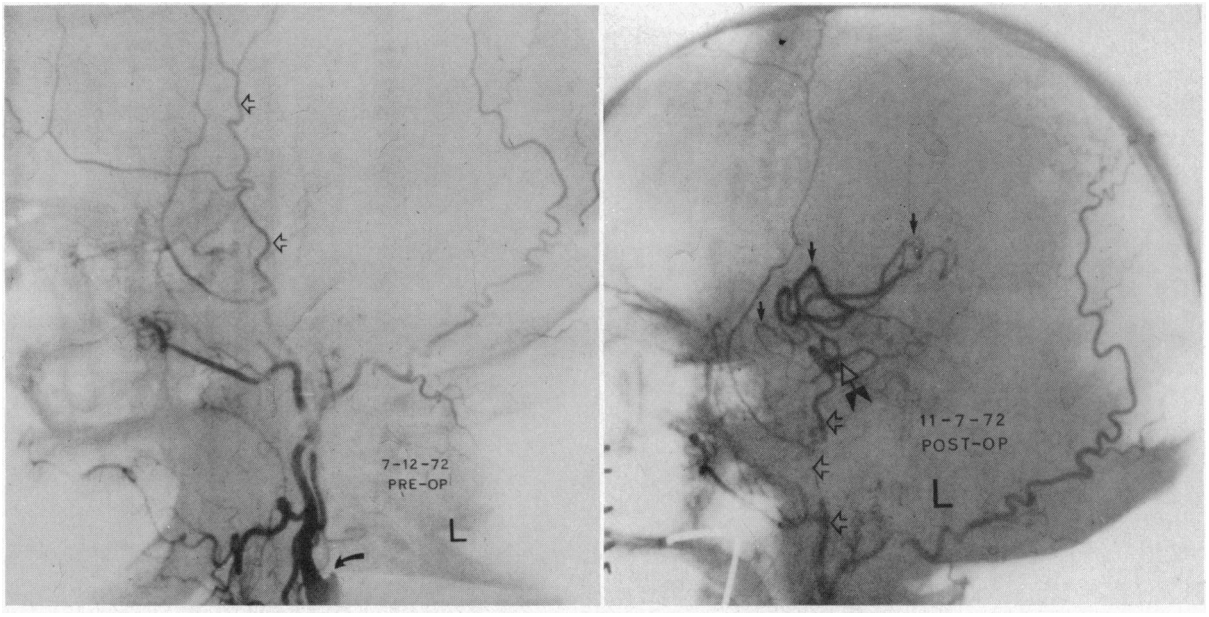

Figure 3.—Left, lateral view of preoperative left common carotid arteriogram showing complete occlusion of left internal carotid artery at the bifurcation (black curved arrow). The open arrows point to superficial temporal artery. **Right,** postoperative (four months) left external carotid arteriogram shows slight hypertrophy of left superficial temporal artery (open arrows), point of anastomosis (black and white arrow), and middle cerebral artery filling (black arrows).

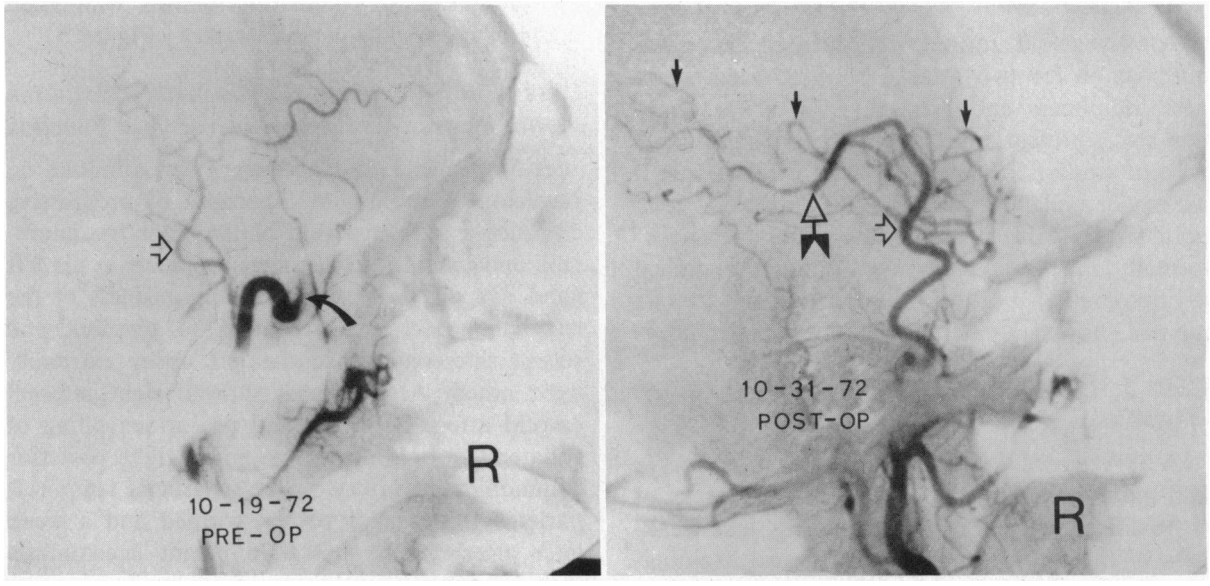

Figure 4.—Left, preoperative right common carotid arteriogram, lateral view, shows complete occlusion of right internal carotid artery at the carotid siphon (curved black arrow). Open arrow points to preoperative right superficial temporal artery. **Right,** right external carotid arteriogram, lateral view, one week postanastomosis of right superficial temporal artery and right middle cortical artery branch. Note the dilated right superficial temporal artery (open arrow). Point of anastomosis (black and white arrow). Small black arrows indicate filling of the right middle cerebral artery branches.

attack of complete right hemiplegia and aphasia. Complete motor aphasia and total flaccid right hemiparesis were noted on physical examination. Arteriograms revealed complete occlusion of the left internal carotid artery. Superficial temporal artery bypass was performed 20 days after onset of the severe deficit. Four months later the patient had residual very mild clumsiness of the right hand and minimal dysphasia. Doppler flow studies showed the anastomosis to be patent.

A post-operative arteriogram shows a patent, well-functioning anastomosis, with enlargement of the superficial temporal artery.

Discussion

Historically, the first two surgical procedures of this type (superficial temporal artery bypass) were performed for cerebro-vascular insufficiency almost simultaneously, one in Burlington, Vermont, (Donaghy) and one in Zurich, Switzerland (Yasargil) in late 1967.[23] The anastomosis site in the Zurich case continues to be open and enlarging, indicating that long term patency is possible. The patient has remained asymptomatic.[24]

Since 1967 about a hundred such procedures have been performed in the world. At the European Congress in Prague in 1971, reports on 44 temporal bypass operations and postoperative results were presented, and the patency rate was approximately 60 percent.[25] Of 28 such operations performed by Yasargil and reviewed by one of us (Chater) in 1971, the mortality rate was zero and the morbidity was very low. The patency rate of the anastomotic sites was 66 percent and was associated with a similar percentage of clinical improvement.[24] Twelve such operations have been performed at this hospital to date. In six cases in which postoperative arteriograms have been done the anastomosis is patent. Eight of the 12 patients have shown significant clinical improvement. One patient died a month after operation after acute bleeding from a chronic gastric ulcer. (Permission for autopsy was not obtained.) In three patients there has been no clinical change.

Indications

The tentative indications for such a collateral augmentation procedure may be outlined as follows:

1. *Multiple vessel occlusions associated with generalized low cerebral perfusion symptoms,* such as, mental deterioration and memory loss, vertigo, ataxia, and light-headedness.[26]

2. *Focal symptoms and signs resulting from inadequate regional perfusion.*

Types:

(a) Internal carotid artery occlusion with inadequate collaterals (Figures 3 and 4).

(b) Internal carotid artery siphon stenosis (inaccessible) with inadequate collaterals (Figure 2).

(c) Middle cerebral artery occlusion with inadequate collaterals and mild neurologic deficit.

(d) Middle cerebral artery stenosis with inadequate collaterals (Case 2 of series).

(e) Vertebral-basilar arterial stenotic lesions (inaccessible) with inadequate collaterals.

This collateral augmentation approach may also be useful in the therapy of non-atherosclerotic occlusive processes in the cerebrovascular systems, such as, arteritis.[27-31]

REFERENCES

1. The President's Commission on Heart Disease, Cancer, and Stroke. US Government, Washington, DC, 1964

2. Ostfeld, AR: Are strokes preventable? Med Cl of NA, 51: 105-111, Jan 1967

3. Karpman HL, Kalb IM, Sheppard JJ: The use of thermography in a health care system for stroke. Geriatrics 27:96, Feb 1972

4. Fields WS, North RR, Hass WK, et al: Joint study of extracranial arterial occlusion as a cause of stroke. JAMA 203:955-960, Mar 11, 1968

5. Gordon EE, Kohn KH: Evaluation of rehabilitation methods in the hemiplegic patient. J Chron Dis 19:3-16, 1966

6. Marshall J: The natural history of transient ischaemic cerebrovascular attacks. Quart J Med, 33:309-324, Jul 1964

7. Acheson J, Hutchison EC: Observations on the natural history of transient cerebral ischaemia. Lancet ii, 871-874, 1964

8. Friedman GD, Wilson WS, Mosier JM, et al: Trasient ischemic attacks in a community. JAMA 210:1428-1434, 1969

9. Marshall J: Angiography in the investigation of ischemic episodes in the territory of the internal carotid artery. Lancet 1:719-721, 1971

10. Hass WK, Fields WS, North RR, et al: Joint study of extracranial arterial occlusion—II. Arteriography, techniques, sites and complications. JAMA 203:961-968, 1968

11. Blaisdell WF, Clauss RH, Galbraith JG, et al: Joint study of extracranial arterial occlusion—IV. A review of surgical considerations. JAMA 209, 12, 1969

12. Blaisdell WF, Hall AD, Thomas AM, et al: Cerebrovascular occlusive disease—Experience with panarteriography in 300 consecutive cases. Calif Med 103, 5:321-322, Nov 1965

13. Wylie EJ, Hein MF, Adams JE: Intracranial hemorrhage following revascularization for treatment of acute stroke. J Neurosurg 21:212-215, Mar 1964

14. Tonnemacher K: Unpublished data

15. Young W, Gofman JW, Tandy R: Quantitating of atherosclerosis—I. Relationship to artery size. Am J Cardiol, 6:288, 1960

16. Resch JA, Loewenson RB, Baker AB: Physical factors in the pathogenesis of cerebral atherosclerosis. Stroke Vol 1, No 2: 77, Mar-Apr 1970

17. Diethrich EB, Liddicoat JE, McCutchen JJ, et al: Surgical significance of the external carotid artery in the treatment of cerebrovascular insufficiency. J Cardiov Surg 9:213, May-Jun 1968

18. Fields WS, Bruetman ME, Weibel J: Collateral circulation of the brain—2, 3: Monographs in the Surgical Sciences. Baltimore, Williams & Wilkins Co, 1965

19. De Bakey ME: Concepts underlying surgical treatment of cerebrovascular insufficiency. Clin Neurosurg 10:310-340, 1964

20. De Bakey ME, Crawford ES, Cooley DA, et al: Cerebral arterial insufficiency—One to 11-year results following arterial reconstructive operation. Ann Surg 161:921-945, 1965

21. Romanus FCA, Abramowicz A: Changes in brain and pial vessels in arterial border zones. Arch Neurol 11:40-48 Jul 1964

22. Hossman KA, Sato K: Recovery of neuronal function after prolonged cerebral ischemia. Science 168-375, 1970

23. Yasargil MG, Krayenbuhl JA, Jacobson JH II: Micro-neurosurgical arterial reconstruction. Surgery 67, 1:221-233, Jan 1970

24. Chater NL, Yasargil MG: Results of temporal artery bypass procedures in the treatment of cerebrovascular disease. Presented at Congress of Neurological Surgeons, Miami, Florida, Oct 1971

25. Donaghy RM: What's new in surgery? Surg Gynec & Obstet 134:369, Feb 1972

26. Wylie EJ, Ehrenfeld WK: Extracranial Occlusive Cerebro-Vascular Disease—Diagnosis and Management. Philadelphia, W. B. Saunders, 1970, chap 7

27. Levine J, Swanson PD: Non-atherosclerotic causes of stroke. Ann Int Med 70:807-816, Apr 1969

28. Shillito J: Carotid arteritis—A cause of hemiplegia in childhood. J Neurosurg 21:540, 1964

29. Tavaras: Multiple progressive intracranial arterial occlusions: A syndrome of children and young adults. Am J Roentg ii, 106: 235-267, Jun 1969

30. Seneviratne BIB, Ameratunga B: Strokes in young adults. Br Med J 2:791-793, Sep 1972

31. DeVico DC: Vertebrobasilar occlusive disease in children. Arch Neurol 26:278-281, Mar 1972